OT DISPERSION AND EARLY ARRHYTHMIC RISK IN ACUTE MYOCARDIAL INFARCTION

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CERTIFICATE

This is to certify that this dissertation entitled," QT dispersion and early arrhythmic risk in Acute Myocardial Infarction" is the bonafide record work done by Dr.J.R.S.VIJAYBABU SATHISHKUMAR , under our guidance and supervision in the Department of Medicine, Thanjavur Medical College, Thanjavur, submitted as partial fulfillment for the requirements of M.D., Degree Examination Branch I, GENERAL MEDICINE MARCH 2007, under The Dr. M.G.R. Medical University, Chennai.

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CONTENTS

INTRODUCTION

Over 200 years have elapsed since William Heberden's[1768],one sentence description of angina pectoris before the Royal college of Physicians-

"Those , whose are afflicted with it, are seized, while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast, which seems as if it would take their life away, if it were to increase or to continue; the moment they stand still, all this uneasiness vanishes", this has been repeatedly confirmed but seldom improved upon.

Until 1921, it was believed that acute myocardial infarction was uniformly fatal. It was James Herrick, a Chicago physician, who first described survival after infarction in an article that appeared in the journal of the American Medical Association in 1921.

In subsequent years, as the understanding of the pathophysiological principles of myocardial infarction begin to grow, as did the treatment modalities and the prognosis began to improve. Despite impressive studies in diagnosis and management over the past 3 decades, AMI continues to be a major public health problem in industrialized world and is becoming an increasingly important problem in developing countries.

Modern 'reperfusion era' of coronary care was introduced by intracoronary and intravenous thrombolysis , increased use of aspirin and development of PTCA and intracoronary stents for AMI. The transition of coronary care from

pathophysiologically based decision making to “evidence-based decision” making is supported by the rich database of clinical trials and meta-analysis.

If subjects at high risk of sudden cardiac death were easily identifiable, then targeted therapy might be able to reduce cardiac deaths. Unfortunately, we do not yet possess an applicable screening method for this purpose. Techniques exists for this such as signal-averaged electrocardiography, T-wave alternans and heart rate variability, but they have variable success and tend to require specialized equipment, making them difficult in routine practice. Another possibility is QT interval analysis, which stems from the fact that individuals with long QT syndromes are known to be at high risk of sudden cardiac death.

Taking this principle one step further, it is possible that the variation of QT intervals within an ECG in more routine patients may also contain prognostic information. ‘QT interval dispersion’ is at present undergoing vigorous assessment for this purpose. Several years ago, Campbell et al –[1] enthusiastically called it the “ electrophysiological Holy Grail ”. The number of studies indexed in the Medline on QT dispersion has risen 34-fold since its description in 1990.

In this study, an attempt has been made to find out QT dispersion in healthy individuals and patients of Acute Myocardial Infarction and to find out correlation, if any, between QT dispersion and the incidence of ventricular arrhythmias in acute myocardial infarction.

AIMS OF THE STUDY

This prospectively designed study aimed to examine

1. QT dispersion in normal individuals.
2. QT dispersion in AMI.
3. QT dispersion and site of infarct.
4. QT dispersion and reperfusion therapy.
5. QT dispersion, ventricular arrhythmias and mortality in AMI.

REVIEW OF LITERATURE

HISTORICAL ASPECTS:

A Dutch physician, William Einthoven [1860-1927] introduced the ECG 'PQRST' designations we use today, and the QT interval has been known since 1887 to represent ventricular electrical activities-[4] . One hundred years later, a group from Newcastle –[5] proposed that the interlead QT interval differences within a 12 lead ECG might reflect regional differences in myocardial refractoriness, and that this might predict cardiac dysrhythmias. Animal –[6] and human –[7] studies supported this observation. Using epicardial monophasic action potentials in isolated rabbit hearts, Zabel et al –[8] correlated this QT interval variation with the degree of homogeneity in ventricular repolarisation. This correlation would suggest that QT dispersion is at best a surrogate marker, rather than an accurate measure of ventricular repolarisation . In reality, the repolarisation process is nondipolar and hence there is only one end-of-repolarisation and the onset of repolarisation is nearer to the T-wave peak within a surface ECG-[9].

The view that sudden obstruction of a coronary artery was incompatible with life for more than few minutes persisted even in twentieth century. The modern era can be said to have begun with the autopsy studies of 'Herrick', who concluded in 1912, that the clinical syndrome of myocardial infarction results from the acute thrombolytic occlusion of coronary arteries, with resulting down stream necrosis-[10]. His statement , " Death results in nearly all these cases, yet it may be delayed for

many days. More than this, there is, as has been shown by reference to experimental work, no intrinsic reason why some patients with obstruction may not recover-[10].

Since then management of myocardial infarction has undergone numerous changes with progressive reduction in mortality from the pre-ICU era to ICU era to the recent thrombolytic era.

EPIDEMIOLOGY

Acute myocardial infarction is one of the most common diagnosis in hospitalized patients in industrialized countries.

Approximately 1.1 million AMI occur each year in U.S. and mortality is upto 30%.

INDIAN SCENARIO:

- While CAD has been halved in west in the past 50 yrs, rates have doubled in India.
- CAD prevalence in Urban India – 10%

Rural India – 5% [12-14].

PECULIARITIES IN INDIAN CAD:

1. Extreme prematurity. CAD mortality in Indians< 30 yrs of age is 3 fold higher than whites[15].
2. Women have rates similar to men despite smoking being uncommon in Indian women[16].
3. Higher prevalence of glucose intolerance and lower prevalence of conventional coronary risk factors[17].

Although atherosclerosis is the most common cause of luminal narrowing in coronary artery disease, multiple non-atherosclerotic causes account for 4-7% of causes of acute MI[18-19].

PATHOGENESIS AND PATHOLOGY

Coronary Atherosclerosis[AS] in the form of stenosing plaques is the major etiologic factor behind various clinical syndromes of IHD. Only in a small minority is non-atherosclerotic lesion like emboli, vasculitis,dissection may be the cause.

Stenosis caused by atheromatous plaque may be eccentric or concentric[20]. Eccentric plaque causes total variation in lumen leading to variable luminal stenosis. Concentric plaque has fixed stenosis[21].

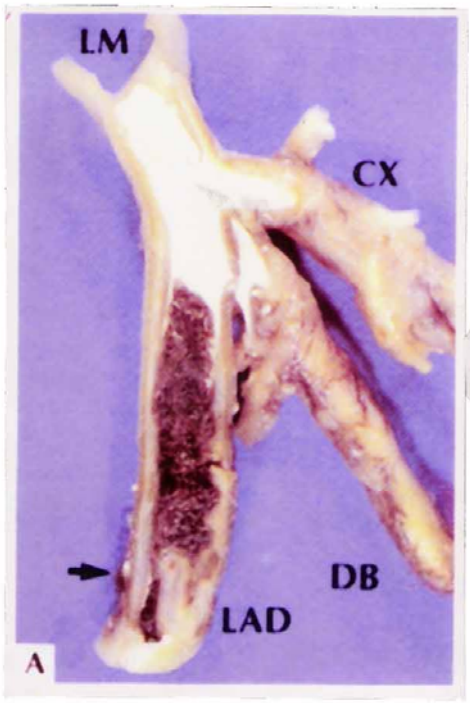
Fatty streaks – earliest lesion in AS can be found in coronaries of children[22] . With increasing age fatty streaks evolve into fibromuscular plaques. Platelet adherence, platelet aggregation and release of smooth muscle growth factors, causes embryonic atherosclerotic plaque to increase in size.

Smooth muscle and fibrous tissue growth factors are released from disturbed endothelium and macrophages derived from adherent monocytes. Abnormal lipid transport from arterial lumen, necrosis and calcification occur in AS plaque.

Clinical presentation and prognosis of coronary AS depend on the plaque type rather than the size[23]. Type IV and Va plaques are plaques prone for disruption.

PLAQUE MORPHOLOGY[24]:

- ❖ Stable plaque – stable angina
    - Small lipid pool.
    - Thick fibrous cap.
    - High grade stenosis.
    - Not at bend or branch.



L.S. of proximal LAD showing ruptured plaque wi
red thrombus propagating upto first Diagonal bra

L.S. of LAD showing gray white occluding thrombus.

- ❖ Unstable / vulnerable plaque
    - Large lipid pool.
    - Younger, less stenotic.
    - Located at branch points.
    - Thin plaque cap.
    - Numerous macrophages.
    - Paucity of smooth muscle cells in cap.

MECHANISM OF THROMBOSIS:

Three forms of vascular injury: IP et al[25].

1. Type I – Endothelial dysfunction.
2. Type II – Endothelial denudation and intimal damage with intact internal elastic lamina.
3. Type III – Endothelial denudation and damage of both intima and media.

Rupture of plaque is a function of internal plaque changes[Intrinsic factor] and external stresses[Triggers]

Internal – plaque vulnerability.

External – systemic Triggers- Exercise

Cigarette smoking

BP swings

	Epinephrine
	Emotions
	Assuming erect posture
	Cold exposures.
Local Triggers	Infection
	Inflammation[26].

Platelet activation with release of thromboxane A_2 is an accompanying phenomenon – Hrish et al[27].

BIOCHEMISTRY AND PATHOLOGY

PATHOLOGY:

Persistent myocardial ischemia leads to infarction with wave front progressing from subendocardium to sub-epicardium.

Myocyte necrosis – 3 types

1. Coagulation necrosis – "Atonic Death" contractile ability of myocyte is lost.
2. Contraction band necrosis – "Tetanic Death" Occurs at periphery of infarct. Hyper contractile state.

3. Colliquative myocytolysis – “Failing Death” Vacuolated myocyte, loose their function.

HISTOLOGICAL CHANGES:

➢ P.M.N.s invade tissue	6 – 8 hours.
➢ Cross structures of myocyte lost	24 hours
➢ Sarcoplasm assumes granular appearance	
➢ Macrophages infiltration in lesion	48 – 72 hours
➢ Neocapillarization begins with fibroblast and capillaries	3 – 5 days
➢ Collagen deposition starts	7 – 10 days
➢ Leads to fibrous scar.	

BIOCHEMISTRY AND ULTRASTRUCTURE:

20 – 40 minutes of myocardial ischemia leads to irreversible cellular derangement.

- Sarcolemmal injury is the critical event determining the irreversibility of cellular damage, leads to leakage of essential enzymes out of cell and entrance of calcium and water into the cell.
- Mitochondria swollen with amorphous matrix.
- High energy phosphates[ATP] rapidly depleted[28].
- Anaerobic glycolysis employed initially with glucose as substrate and large amount of intracellular lactate accumulation[29].
- Arachidonic acid liberation - FFA ↑.
- O_2 derived free radicals and intracellular calcium overload, 'chokes' the mitochondria[30,31].

CIRCADIAN VARIATION:

AMI has marked circadian periodicity with peak prevalence between 6A.M. and noon. There is 3- fold increase in frequency of infarction at peak 9.00 A.M.[32]. This is due to diurnal variation in thrombotic tendencies – platelet aggregability[33], circadian variations in blood pressure[34] and sympathetic nervous system[35].

RISK FACTORS FOR AS/ AMI

NON-MODIFIABLE RISK FACTOR:

- ✓ Age.
- ✓ Male gender.
- ✓ Low socio economic status.
- ✓ Family history of early onset CAD.

MODIFIABLE RISK FACTORS:

CATEGORY I:

- ✓ LDL cholesterol ↑
- ✓ HDL cholesterol ↓
- ✓ Atherogenic diet
- ✓ Cigarette smoking
- ✓ Hypertension
- ✓ LV hypertrophy
- ✓ Thrombogenic factors.

CATEGORY II:

- ✓ Diabetes mellitus
- ✓ Physical inactivity
- ✓ Triglycerides
- ✓ Obesity

CATEGORY III:

- ✓ Phycosocial factors
- ✓ Lipoprotein(a)
- ✓ Homocysteine
- ✓ Inflammation marker – particularly CRP
- ✓ No alcohol consumption
- ✓ Oxidative stress
- ✓ Post menopausal status.

INDIAN SCENARIO[37]:

- Higher prevalence of Diabetes , Insulin resistance syndromes, central obesity.
- Lower prevalence of conventional risk factors like Hypertension, Obesity, Cigarette smoking, high cholesterol.
- Emerging newer risk factors like high lipoprotein(a), homocysteine, Apo B, triglycerides, fibrinogen, PAI-I.
- Low HDL and HDL_{2b}.
- Small dense LDL.

HISTORY AND PHYSICAL EXAMINATION FOR AMI

Classic symptoms – chest discomfort which is retrosternal (or) precordial, described as pressure, aching, burning, crushing, squeezing, heavy, swelling or bursting in quality. Radiation to arm (medial aspect), neck or jaw, back etc.[38].

Skip areas with retrosternal pain common.

Approximately 23% of MI go unrecognized due to atypical symptoms or presentations[39].

- Nausea or vomiting / Indigestion(common with IWMI)
- Atypical location of pain – arm, back, jaw, occiput
- Profound fatigue of rapid onset
- Cerebral or peripheral embolus
- Pulmonary edema of sudden onset
- Mental obtundation
- Severe ventricular dysrhythmias
- Painless myocardial infarction is common in elderly >60 yrs and diabetics.

CLINICAL EXAMINATION:

Findings vary from normal to shock from low cardiac output.

1. Rhythm: varies from regular with tachycardia and a few extrasystoles to multiple ectopics, VT, and VF. Atrial fibrillation is usually unimportant. Acute MI involving inferior wall is often associated with transient hypo tension and sinus bradycardia.

Bezold Jarish Reflex – Bradycardia

Hypo tension

Nausea / vomiting.

stimulation of inhibitory cardiac receptors in the inferoposterior wall of LV.

2. Blood pressure is often elevated at first (from emotion) and later falls.
3. Jugular venous pressure is normal, unless there is complicating Heart failure.

PRECORDIAL PALPATION:

- Diffuse apical impulse
- Dyskinesia – paradoxical bulge in late systole.
- Palpable S_4
- Paradoxical splitting, rarely due to severe LV dysfunction.

AUSCULTATION:

- Muffled heart sounds
- Muffled S_1 common in IWMI due to prolonged PR interval.
- S_4 – 98% of cases of AMI[40].
- S_3 – 15 – 20% of cases of AMI[41.]
- Pericardial rub
- Crescendo – decrescendo mid systolic murmur due to ischemia of papillary muscles.
- Mitral regurgitation murmur.
- PSM due to ventricular septal rupture.

EXAMINATION OF LUNG FIELDS:

Pulmonary congestion occur in 30 – 40 % of uncomplicated MI.

KILLIP AND KIMBALL CLASSIFICATION:

- Class I – No pulmonary rales or S_3
- Class II – Bibasilar rales that persist after coughing.

  < 50 % of lung fields or S_3

- Class III – Rales over one-half of the lung fields bilaterally with radiographic evidence for pulmonary edema (> 50 % of lung fields)
- Class IV – Cardiogenic shock.

FORRESTER STAGING:

- I - Normal hemodynamics.
- II – Hyperkinetic state.
- III – Hypovolemia
- IV - LV failure – A. Mild

  B. Severe

- V - Cardiogenic shock.
- VI – Shock due to RVMI.

DIAGNOSIS OF MYOCARDIAL INFARCTION:

Triad of

i. Chest pain

ii. Electrocardiographic changes

iii. Plasma enzyme activity.

ELECTROCARDIOGRAPHIC CHANGES OF AMI:

The ECG is a cornerstone in the diagnosis of acute and chronic heart disease. In AMI, typically there are changes in the S-T segments, T wave and QRS complexes. It is only the QRS changes which are diagnostic of myocardial infarction.

THE QRS CHANGES OF AMI:

Two QRS abnormalities may be indicative of myocardial infarction.

They are

1. Localized, inappropriately low, R wave voltage.
2. Abnormal Q waves.

The development of a negative wave (a Q wave) and the reduction in size of the positive wave are the result of loss of positivity from necrosis of myocardium.

LOSS OF R WAVE VOLTAGE:

If infarction involves only part of the thickness of the myocardial wall the QRS complexes recorded from the area of the infarction will show a reduction on R wave voltage.

ABNORMAL Q WAVES AND QS COMPLEXES:

When infarction involves the full thickness of the myocardium there will be total loss of R waves in leads overlying the infarcted zone i .e. the waves will be entirely negative (QS complexes). These negative waves are the result of depolarization of the posterior wall of the ventricles traveling from the endocardium to epicardium (and therefore away from the precordial leads). The four possible QRS changes which may indicate the presence of myocardial infarction are as follows:

1. Reduced R wave voltage (where it can confidently be ascertained that this has occurred).
2. Abnormal Q waves without any conclusive evidence of R wave reduction.
3. Abnormal Q waves with evidence of reduced R wave voltage.
4. Abnormal QS complexes.

A normal q wave is not > one-quarter of the height of the ensuing R wave and is < 0.04 s in duration. Abnormal Q waves have a depth > 25 % of the height of the ensuing R wave, or a duration $\supseteq$ 0.04 s.

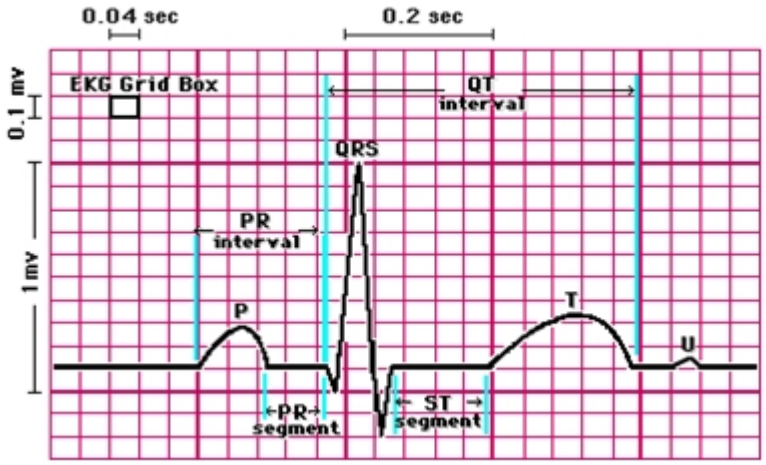
THE ST-T SEGMENT CHANGES OF INFARCTION:

In the early stages of infarction S-T segment elevation usually occurs and may occasionally be dramatic in degree. Abnormal S-T segment elevation occurs in leads facing an area of transmural infarction. Leads looking at the heart from the opposite aspect will show 'reciprocal' S-T segment depression at a time when there is 'primary' S-T segment elevation in the leads related to the infarction. The precordial leads and lead I and aVL on the one hand and the inferior limb leads (leads II, III, and aVF) on the other hand are mutually reciprocal in this aspect.

THE T WAVE CHANGES OF INFARCTION:

A whole variety of non-specific T wave changes may occur in association with myocardial infarction. These include flattening of the T waves, inversion of T waves and abnormally tall T waves. Widespread deep symmetrical T wave inversion, whilst still, strictly speaking a non-specific change, suggests subendocardial infarction.

THE QT INTERVAL:



The interval from the beginning of QRS complex to the end of T wave is called QT interval. It is sum total of the time taken by depolarization and repolarisation of the ventricles. The total electrical activity of the ventricles is reflected in QT interval. It is variable in different parts of the ventricles, but the QT interval measured on the surface electrocardiogram represents the total longest interval of QT.

The QT interval shortens with tachycardia and lengthens during bradycardia. In other words, the QT changes with change in RR interval; the latter represents the heart rate. Therefore, for meaningful expression, the QT must be corrected for heart rate called corrected QT (QTc) interval. The QT interval is corrected for what it would be at a rate of 60 bpm.

$$\text{Corrected QT (QTc)} = \text{QT interval in sec} \;/\; \sqrt{\text{RR interval in sec}}$$

By Bazett's formula.

MEASUREMENT OF QT:

It is measured in seconds from the beginning of Q wave to the end of the T wave. It is difficult to measure sometimes because it is difficult to delineate the beginning and end of the interval. It is customary to measure it in the lead having 'q' wave such as leads I, II, aVL, V5 or V6 so as to avoid error of ignoring the initial part of QRS. Similarly, the end point of T wave may be difficult to find out, but if U wave is present, then it becomes easy as the dip between T and U wave becomes the end point of T wave.

Sleep prolonged the QT interval by 18 ms at a heart rate of 60 beats / min and by 2 ms at a heart rate of 50 beats / min compared with the waking state[42]. This diurnal variation is thought to be related to autonomic tone.

CAUSES OF LONG QT INTERVAL:

- Physiological – during sleep
- Congenital-prolonged QT syndrome – The Jervell-Lange-Nielsen syndrome and Romano-Ward syndrome.
- Acquired – Hypocalcemia
- Acute myocarditis
- Acute myocardial infarction
- Quinine, Quinidine and Procainamide effect
- Antidepressants (Tricyclic and Tetracyclics)
- Head injury with intracerebral bleed
- Hypothermia

- Idiopathic hypertropic cardiomyopathy
- Advanced or complete heart block
- Ventricular tachycardia – Torsade de pointes.

SHORT QT INTERVAL:

- Digitalis effect
- Hyperthermia
- Vagal stimulation
- Hypercalcemia.

QT DISPERSION(QT d):

Day et al[43] first proposed that interlead variability of QT interval in 12-lead electrocardiogram – QT dispersion [defined as the difference between maximum and minimum QT interval duration] reflects dispersion of ventricular recovery time.

Theoretically the QT value should be the same but differences occur because of different projections of the ventricular complex on the limb axis in different leads. For instance, when the vectors are perpendicular to the QRST axis an isoelectric segment may be recorded at the onset of the QRS complex, at the end of the T wave thereby causing artificial shortening of the QT interval. In the precordial leads differences in the QT interval may be due to differences in the repolarisation duration at the site facing the recording electrodes. The latter factors probably account for most of the so called QT dispersion.

The QT interval is measured from the beginning of QRS to the end of T wave (i.e. return to the TP baseline). If U waves are present, then QT interval is measured to the nadir of the curve between the T and the U waves. For calculation of QT d, 3 consecutive cycles are measured in each lead and the mean of these 3 cycles is taken as mean QT c of that lead. The QT may be corrected by Bazett's formula. The mean normal QTcd is 45±15 ms.

CLINICAL CONDITIONS ASSOCIATED WITH WIDE QTd INTERVAL:

- Congenital or acquired prolonged QT syndrome
- Hypertropic cardiomyopathy
- Mitral valve prolapse
- Coronary artery disease.

CLINICAL SIGNIFICANCE:

- QT d is believed to be a measure of electrical inhomogenity in the heart that decreases an individual's threshold for ventricular arrhythmias, hence greater the QTd , greater is the chance to develop an arrhythmia.
- Heart rate, reflex vagal activity and cardiac afterload physiologically alter the QT dispersion.
- A significant QT dispersion was found both in patients with congenital and acquired long QT intervals, which is presumed to be the cause of Torsade-de-pointes in these conditions.
- Certain antiarrhythmic drugs decrease the QT dispersion hence, QT dispersion may be used to evaluate the efficacy of the drug.

- Practical utility as a marker of acute ischemia with atrial pacing.

PLASMA MARKERS OF MI:

As the myocyte become necrotic, the integrity of the sarcolemmal membrane is compromised and intracellular macromolecules (cardiac markers) begin to diffuse into the cardiac interstitium and ultimately into the microvascular and lymphatics in the region of the infarct. Molecular markers used or proposed – for use in the diagnosis of Acute Myocardial Infarction[44].

Marker	Time to initial elevation(hrs)	Mean time to peak(hrs)	Time to return to normal(hrs)	Sampling schedule
HFABP heart fatty acid binding proteins	1 – 5	5 – 10	24	On presentation,then 4 hrs later
Myoglobin	1 – 4	6 – 7	24	1 – 2 hrs after chest pain
MLC (Myosin Light Chain)	6 – 12	2 – 4 days	6 – 12 days	12 hrs after chest pain
cTn I (Troponin I)	3 – 12	24 hrs	5 – 10 days	12 hrs after chest pain
c Tn T (Troponin T)	3 – 12	12 hrs – 2 days	5 – 14 days	12 hrs after chest pain
MB – CK	3 – 12	24	48 – 72 hrs	Every 12 hrs × 3
MB – CK tissue isoform	2 – 6	18	-	60 – 90 mins after chest pain
Enolase	6 – 10	24	48	Every 12 hrs×3
LDH	10	24 – 48	10 – 14 days	24 hrs after chest pain
MHC (Myosin	48	5 – 6 days	14 days	After 2 days of chest pain

Heavy Chain)				

ENZYMATIC CRITERIA FOR DIAGNOSIS OF MI[45]:

- Serial increase, then decrease of plasma MB-CK, with a change > 25 % between any 2 values
- MB-CK >10 – 13 U/L or > 5 % of total CK activity.
- Increase in MB-CK activity > 50% between any 2 samples, separated by at least 4 hrs.
- If only a single sample available, MB-CK elevation > 2 fold.
- Beyond 72 hrs, an elevation of Troponin T or I or LDH_1>LDH_2.

Early diagnosis of MI is offered by MB-CK subforms and Myoglobin of which MB-CK subform are most specific.

- Absolute level of MB-CK_2 isoform greater than 1.0 U/L within 4 – 6 hrs of MI.
- CK-MB_2/ CK-MB_1 greater than 2.5 (within 4 – 6 hrs of MI). 91 % specificity and sensitivity[46].

CK ISOENZYMES:

- 15 % of CK in the myocardium is CK-MB, which provides sensitivity and specificity as a marker.
- CK-MB mass/ CK activity >2.5 indicates myocardial rather than skeletal muscle as a source of CK-MB.

TROPONINS:

- Cardiac Troponins are very specific for the myocardial injury. Normal plasma value for Troponin are near 0 and hence they are very sensitive also.
- Troponins are not elevated in CRF.

DIAGNOSIS OF RE-INFARCTION:

Secondary rise in CK-MB, 36 – 48 hrs after onset of symptoms, $\supseteq$ 50 % above the preceding baseline.

UPPER LIMIT OF NORMAL FOR MARKERS[45]:

- Myoglobin 85ng/ml
- CK-MB 9 IU/ L
- $CK\text{-}MB_2$ 1.0 U/L
- $CK\text{-}MB_2/CK\text{-}MB_1$ $\supseteq$2.5
- Troponin T 0.1ng/ml
- Troponin I 1.5ng/ml

OTHER BIOCHEMICAL ALTERATIONS:

- Lipoprotein fractions are relatively unchanged in initial 1 – 2 days but decrease significantly over subsequent days and weeks. Hence lipid measurements are done in 24 to 48 hrs or 6 – 8 weeks later[48].

- ❖ WBC count show mild increase reaching peak in 3 – 5 days.
- ❖ Erythrocyte sedimentation rate – maximum in the second week.

IMAGING TECHNIQUE:

CHEST ROENTGENOGRAM:

Amidst excluding other causes of chest pain such as pneumothorax, pulmonary infarction with effusion, aortic dissection, skeletal fractures, presence of pulmonary edema and increased heart size, a group of patient at high risk[49].

ECHOCARDIOGRAPHY:

Assessing patients with non-diagnostic ECG's. Presence of regional wall motion abnormalities provides strong supportive evidence of acute coronary syndrome, generally transmural or Q – wave Myocardial Infarction[50,51].

RADIONUCLIDE ASSAY:

Role in certain patients with RVMI by showing localized contractile abnormalities, (or) 99_mTc – pyrophosphate uptake[52]. Infarction not diagnosed by standard means, detection of residual ischemia, infarct size, hibernating myocardium, ventricular function are other indications.

<u>MANAGEMENT STRTEGIES:</u>

PREHOSPITAL CARE is being increasingly stressed to achieve a further substantial decrease in the mortality rate, since 40 – 65 % of deaths, from AMI occur

within an hour of onset of symptoms, prior to arrival to hospital , most commonly due to ventricular arrhythmias[53, 54].

STRATEGIES:

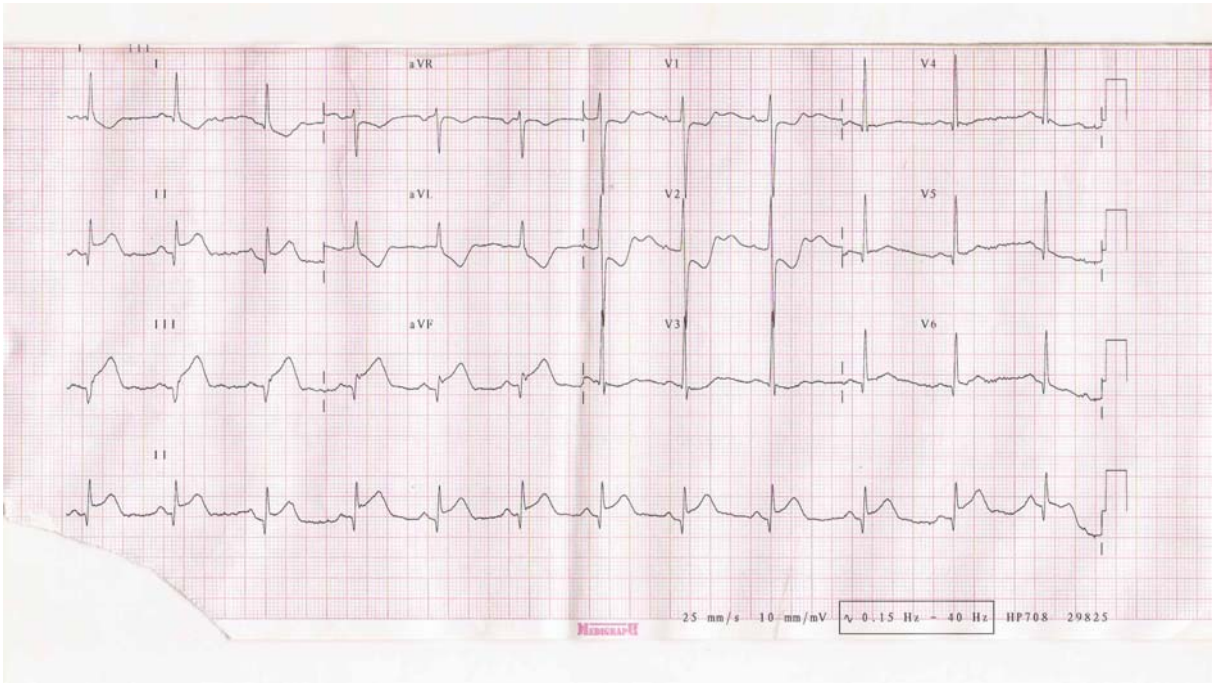
- Education of patients for recognition of symptoms and call for help.
- Role of paramedics in recognition and treating life threatening arrhythmias.
- Pre-hospital thrombolysis – Alteplase for this purpose.

HOSPITAL MANAGEMENT:

DIAGNOSIS AND RISK STRATIFICATION:

- Age, ECG with more number of leads showing ST changes, Q waves, prolonged chest pain.
- SBP < 100 mm Hg and Bibasilar rales indicate high risk.
- Killip classification is useful.

ECG OF A PATIENT WITH INFERIOR WALL MI



INITIAL MANAGEMENT:

OXYGEN ADMINISTRATION:

Hypoxemia is common due to V.P. mismatch. Oxygen administration is reported to decrease ST segment elevation in anterior myocardial infarction[55]. Supplemental oxygen (2 – 4L/ min) for all cases of uncomplicated myocardial infarction with SaO_2 > 90 % after 2 – 3 hrs and extended for those with onset pulmonary congestion and desaturation[56].

ANALGESIA:

To reduce anxiety / pain relief thereby reduce oxygen demand. Narcotics – Morphine (2 – 4 mg). Caution needed in Inferior infarct complicated by RVMI[57].

Nitrates – sublingual Nitroglycerine – 0.4mg doses upto 3 doses on 5 minutes intervals[58]. Decreases oxygen demand. Increases oxygen supply. Caution in Inferior infarct associated with RVMI.

BETA BLOCKERS:

- Pain relief
- Reduce frequency of progression of threatened infarction to complete infarction.
- Reduce life threatening arrhythmias
- Metoprolol – 5 mg every 2 – 5 minutes for total of 3 doses. Oral dose 50 mg 15 minutes after last IV dose, then 50 mg 6 hrly for 48 hrs, then 100 mg every 12h[56].

ASPIRIN:

160 – 325 mg of non-enteric coated chewable aspirin. Inhibits platelet COX – irreversibly and blocks TxA_2 formation, a mediator of platelet aggregation[59]. Aspirin allergy, substituted by Clopidogrel 300mg stratum dose.

REPERFUSION IN AMI:

THROMBOLYSIS:

Thrombolytic therapy reduces the relative risk of in hospital death by 50 % when given in the first hour of onset of symptoms.

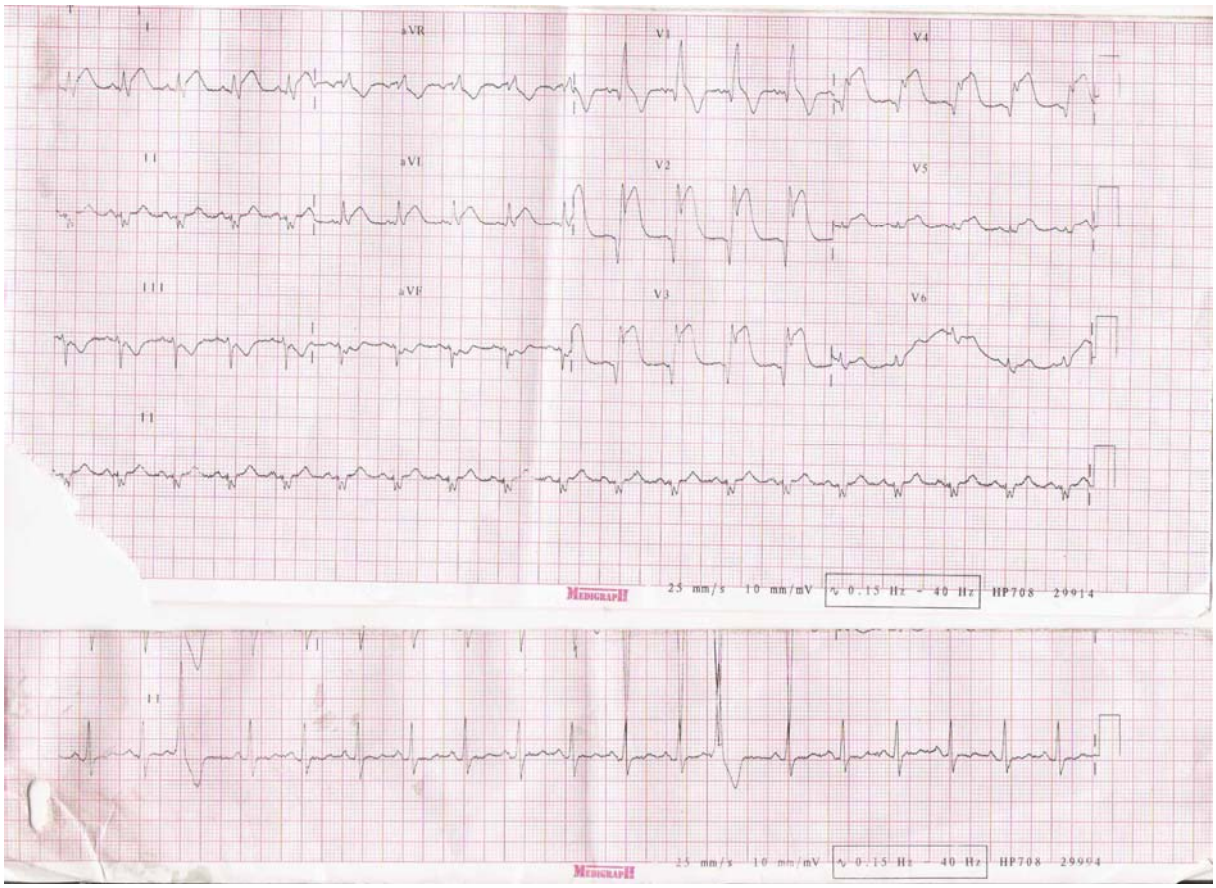
Other uses

- Limits infarct size
- Limit LV dysfunction
- Reduce the incidence of serious complications such as septal rupture, cardiogenic shock and malignant arrhythmias.

Benefit of thrombolysis possible upto 12 hrs, especially if chest discomfort is still present and ST segment remain elevated in ECG.

Goal of thrombolysis is to achieve – TIMI- grade 3, flow in the infarct related artery.

ECG OF A PATIENT WITH ANTERIOR WALL MI



DRUGS USED:

- Streptokinase
- Urokinase
- t-PA – tissue plasminogen activator
- APSAC
- r-PA – recombinant staphylokinase
- TNK- tPA – TNK variant of tissue plasminogen activator
- Lanatoplase

t-PA is more effective than SK in restoring full perfusion (TIMI- grade 3 flow)

- SK – administered in 1.5 million units in a period of 1 hour.
- t-PA – 15 mg bolus followed by 50mg IV, over the first 30 minutes, followed by 35 mg over next 15 minutes.
- Reteplase – double bolus regimen of 10 MU bolus over 2- 3 minutes followed by second 10 MU bolus, 30 minutes later.

Mode of action – conversion of plasminogen to plasmin, which subsequently lyses fibrin thrombi.

CRITERIA FOR THROMBOLYSIS:

- Chest pain consistent with angina
- ECG changes

  ST $\uparrow \supseteq$ 1mm, $\supseteq$ 2 contiguous limb leads.

  ST $\uparrow \supseteq$ 2mm, $\supseteq$ 2 contiguous precordial leads.

  New onset LBBB.

- Absence of contra-indications.

CONTRA-INDICATIONS:

ABSOLUTE:

- Active internal bleeding
- Suspected Aortic dissection
- History of Hemorrhagic cerebrovascular accident or recent non-hemorrhagic C.V.A.
- Recorded B.P. > 200/120
- Trauma or surgery that is a potential bleeding source within previous 2 weeks.
- Intracranial neoplasm or recent head trauma.

RELATIVE CONTRA-INDICATIONS:

- Prolonged , traumatic CPR
- H/O non-hemorrhagic CVA in distant past
- Recent trauma or surgery > 2 weeks previously
- Active peptic ulcer disease
- History of severe Hypertension with DBP >100
- Bleeding diathesis or concurrent use of anticoagulants.
- Pregnancy

COMPLICATIONS:

- Allergic reactions common with SK in 2 % of patients. Minor degrees of hypotension in 4 – 10 % of patients.
- Hemorrhagic stroke is the most serious complication in approximately 0.5 – 0.9 % of patients more with age > 70 years.(Hemorrhage with t-PA higher than SK).

ASSESSMENT OF ADEQUACY OF THROMBOLYSIS:

- Resolution of chest pain
- ST segment resolution in ECG > 50%
- Enzyme kinetics.
- Troponin T or CK-MB mars – post / pre thrombolysis ratio > 5 at 6 minutes >10 at 90 minutes.
- Myocardial contrast Echocardiography.

OTHER MODALITIES OF REPERFUSION:

- Primary PTCA – with or without stenting.
- Rescue angioplasty of failure of reperfusion beyond 90 minutes of thrombolysis.
- Elective PCI in coronary artery re-occlusion or recurrent ischemia.

ANTIPLATELET DRUGS:

GOALS:

- To establish and maintain patency of infarct-related artery
- To reduce the patients tendency to thrombosis and thus the likelihood of mural thrombus formation or deep venous thrombus.

DRUGS:

1. UFH – Unfractionized Heparin. Useful in facilitating thrombolysis and establish patency of IRA in t-PA regimen of thrombolysis. Not so useful in SK regimen.

Dose - Bolus 60U/kg

Maintenance of 12 U/ kg /hr

To keep aPTT – 1.5 to 2 times the control value.

2. LMWH – Low molecular weight Heparins.

Advantage over UFH due to increased anti-factor Xa: IIa ratio, decreased sensitivity to platelet factor IV, as more stable and reliable anticoagulant effect, enhanced bio-availability.

No need for aPTT monitoring.

DRUGS USED:

Nadroparin } efficacy similar to UFH

Dalteparin } efficacy similar to UFH

Enoxaparin superior to UFH

[Enoxaparin 1 mg/ kg subcutaneous every 12 hrs]

GPIIb / IIIa RECEPTOR INHIBITORS:

Facilitates thrombolysis and reduce rate of reocclusion of reperfused vessels.

Mode of action: Block the GP IIb / IIIa receptors of platelets surface which are the final common pathway in platelet aggregation.

DRUGS:

- Abciximab
- Tirofiban
- Eptifibatide

More useful in NSTEMI.

OTHER DRUGS:

1. ACE inhibitors – should be prescribed within 24 hrs of AMI to all patients with MI and overt CHF. Reduction in

ventricular remodeling after infarction with subsequent reduction in CHF.

2. GIK infusion - Glucose – Insulin – Potassium lower concentration of free fatty acids and improves ventricular performance. DOSE: 100 grams of glucose with 20 units of plain insulin and 50 mmol of potassium in 1 litre of water at a rate of 1.5 ml / kg / hr.
3. Magnesium – minimize risk of arrhythmias in high risk patients with MI.

COMPLICATIONS OF AMI:

MECHANICAL COMPLICATIONS:

VENTRICULAR DYSFUNCTION:

After AMI LV undergoes a series of changes in shape, size and thickness in both infarcted and non-infarcted segments, referred to as “ventricular remodeling” , precedes the development of CHF in the months to years after infarction.

Prevention by ACE inhibitors and other vasodilators like nitrates.

CARDIOGENIC SHOCK:

- Most common cause of in-hospital death with myocardial infarction.
- Occurs within hours of onset of infarction due to massive ischemia and necrosis.
- Occurs when 40 % or more of LV is destroyed. Mortality is > 80 %.

CHARACTERISTICS:

- Evidence of organ hypo perfusion – cold clammy skin and extremities, oliguria, decreased mentation.
- SBP < 80 – 90 mm Hg
- LVED pressure > 18 mm Hg
- Cardic index < 1.8 L / M^2

REVERSIBLE CAUSES IN CRDIOGENIC SHOCK:

RVMI, mitral valve rupture, VSR, pulmonary embolism, cardiac tamponade.

Therapy: Dopamine, Dobutamine, Aortic counter pulsation, Mechanical reperfusion.

MITRAL REGURGITATION:

PAPILLARY MUSCLE DYSFUNCTION:

Posteromedial papillary muscle is involved in ischemia / infarction more commonly than anterolateral, since it is supplied predominantly by circumflex artery, whereas later receives dual blood supply.

Presents with sudden onset of apical systolic murmur. Papillary muscle rupture may complicate usually 2 – 7 days after acute MI. Presents as abrupt onset of pulmonary edema, harsh murmur but thrill is unusual. More common in inferior infarct.

VENTRICULAR SEPTAL RUPTURE:

- ✓ 1 – 3 % of AMI is complicated by VSR
- ✓ Majority in the first week
- ✓ New harsh holosystolic murmur along the left sternal border with thrill and sudden clinical deterioration with hypotension and pulmonary congestion.
- ✓ Prompt surgical management is necessary.

FREE WALL RUPTURE[CARDIORRHEXIS]:

- ✓ Occurs in the first week of MI
- ✓ First infarction, history of hypertension, no history of angina pectoris and relatively large Q wave infarct are associated with high incidence of cardiac rupture.
- ✓ Sudden loss of pulse, B.P., and consciousness while ECG continues to show sinus rhythmn[apparent EMD].

RIGHT VENTRICULAR INFARCTION:

- ✓ 40 % of inferoposterior MI have RVMI
- ✓ Spectrum ranges from asymptomatic minimal involvement to major hemodynamic impairment.

CLINICAL FINDING:

CLASSIC – Elevated JVP, systemic hypotension, absence of pulmonary congestion. Additional signs – Kussmaul's sign, cannon A waves in JVP, RV S_3 , S_4 , hepatojugular reflux, T.R. , pulsus paradoxus, right sided pleural effusion.

ECG changes: Diagnostic – ST segment elevation of 1 mm or more in V_4R.

Other changes – decreasing magnitude ST ↑ in precordial leads. ST ↑ in V_2 which is 50% less than the magnitude of ST ↑ in aVF. Leftward shift of transition zones.

COMPLICATIONS: AV block, right to left shunting, TR, cardiogenic shock, and ventricular arrhythmias.

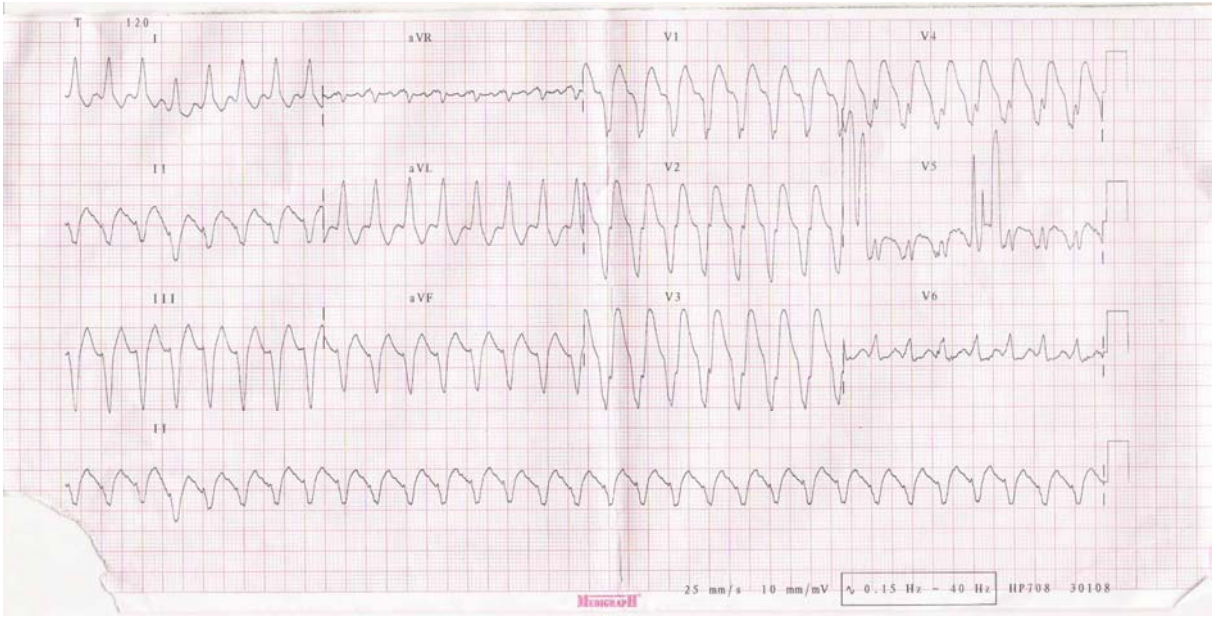
MANAGEMENT:

- ✓ Avoid vasodilators
- ✓ Fluid – 0.9 % NS 1 – 2 L in the first hour, followed by 100 to 200ml / hr to keep SBP- 100 mm Hg or pulmonary artery wedge pressure – 18 cm H_2O.

- ✓ Inotropic therapy – Dobutamine (more beneficial than Dopamine as it does not raise the pulmonary vascular resistance).
- ✓ Reperfusion
- ✓ Maintenance of AV synchrony.

Other complications – Pulmonary embolism, systemic embolism and ventricular aneurysm.

ECG OF A PATIENT WITH VENTRICULAR TACHYCARDIA



<u>ELECTRICAL COMPLICATIONS:</u>

Mechanism for arrhythmias

- ➢ Autonomic nervous system imbalance
- ➢ Electrolyte disturbances
- ➢ Ischemia

- Slowed conduction in the zones of ischemic myocardium.

TACHYARRHYTHMIAS:

Ventricular arrhythmias: VPC's, VT, VF, AIVR.

Ventricular tachycardia is more common in inferior wall MI complicated by RVMI.

Supraventricular arrhythmias – Sinus tachycardia is the most common arrhythmia. SVT, AF and flutter also occur.

Accelerated junctional rhythmn is more common with inferoposterior MI.

BRADYARRHYTHMIAS:

Sinus bradycardia is more common with inferoposterior and RVMI. Managed by Atropine. Persistent bradycardia may need pacing.

AV blocks are particularly common with inferior infarct, attributed to ischemia and enhanced vagal activity.

I degree block needs no treatment

II degree block particularly high grade type II blocks may require pacing.

Complete heart blocks are common in inferior infarct and they are transient, get corrected with reperfusion. Intraventricular conduction abnormalities of which LBBB is more common.

RISK STRATIFICATION AFTER AMI:

Most important predictors of 30 day mortality [Gusto I trial][60]

- Age
- Systolic BP
- Killip class
- Heart rate
- Infarct location

Most important independent predictors of 6 month mortality[GISSI 2 Database][61]

- Ineligibility to perform predischarge exercise test
- Clinical LV failure
- LV dysfunction, defined as EF < 40 %
- Electrical instability
- Age > 70 yrs.

MATERIALS AND METHODS

This study was conducted in the Intensive Coronary Care Unit at Thanjavur Medical College Hospital, Thanjavur, during the period from April 2006 to October 2006.

- One hundred patients of AMI admitted to intensive coronary care unit.
- Both male and female patients were included in the study.
- Both young and old were included in the study.

100 age- and sex matched healthy individuals were included in the study.

INCLUSION CRITERIA:

Patients with a diagnosis of AMI were included in this study. AMI was diagnosed on the basis of

- History of typical chest pain lasting $\supseteq$ 30 minutes
- Unresponsive to nitrates and
- The presence of ST segment elevation in the electrocardiogram of 0.1 mv in $\supseteq$ 2 limb leads or 0.2 mv in $\supseteq$ 2 precordial leads.

EXCLUSION CRITERIA:

Patients were excluded from the study when

- The admission electrocardiogram exhibited technical limitations for analysis of QT dispersion (< 8 evaluable leads)
- Patients were in atrial fibrillation (AF) or flutter.
- Had left or right bundle branch block.
- Patients receiving long term medications with drugs influencing QT duration were also not considered for the study.

METHODOLOGY:

Routine history taking, physical examination and laboratory investigations were performed in all subjects.

Simultaneous 12-lead electrocardiogram was recorded on HEWLETT PACKARD page writer 100 at a paper speed of 25mm/s. QT dispersion was calculated in all the patients of AMI as described by Van de loo et al-[2] on admission, and in those who survived, 24 hours after admission and at the time of discharge from ICCU.

QT dispersion was defined as the difference between the maximum and minimum QT interval measurements among all the measured 12 leads on the standard electrocardiogram-[3] [QT d = QT max – QT min]. For analysis of QT dispersion, RR and QT interval were measured in as many of the 12 leads as possible. Each measurement was taken as the mean value of 2 to 3 consecutive RR and QT intervals.

Ventricular arrhythmias were analyzed and its relationship to QT dispersion was observed.

OBSERVATIONS AND ANALYSIS

This study was done in the Intensive Care Unit of Thanjavur Medical College, Thanjavur during the period from April 2006 to October 2006.

The study was a prospective one.

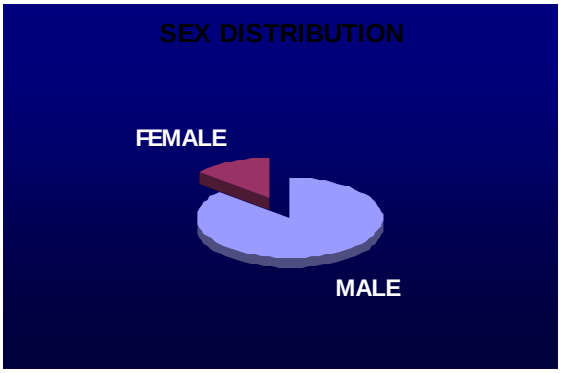
Both male and female patients were taken for the study.

BASELINE CHARACTERISTICS:

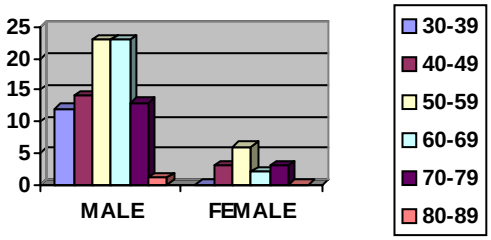
The present study consisted of 100 patients of Acute Myocardial Infarction and an equal number of age- and sex matched healthy individuals. The age of the healthy individuals ranged from 30 to 85 years with a mean of 50.66 ± 17.65 years. The age of patients with AMI ranged from 30 to 82 years with a mean of 55.46 ± 12.97 years. There were 86 males and 14 females in each group. Out of hundred patients of AMI,53 were having anterior wall AMI and 47 had inferior wall AMI.

SEX DISTRIBUTION:

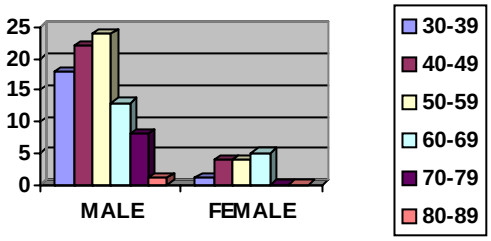
SEX	**NUMBER**	**PERCENTAGE**
MALE	86	86%
FEMALE	14	14%
TOTAL	100	100%

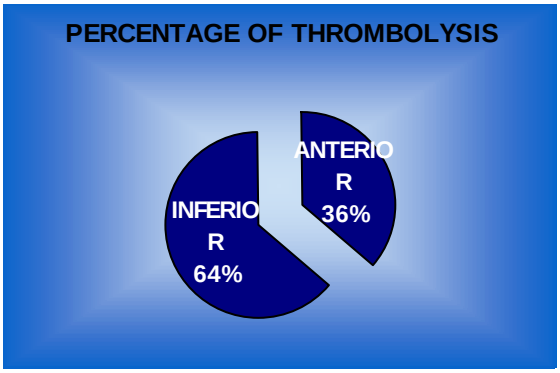


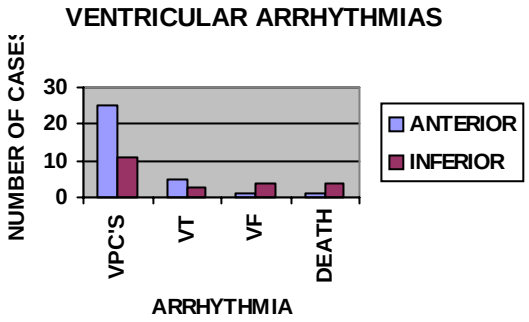
AGE DISTRIBUTION OF AMI CASES

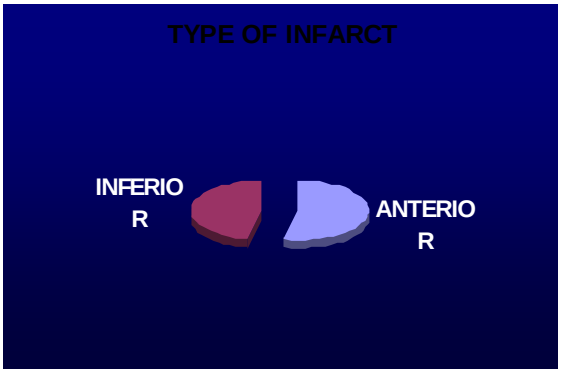


AGE DISTRIBUTION IN HEALTHY INDIVIDUALS




PERCENTAGE OF THROMBOLYSIS
ANTERIOR 36%
INFERIOR 64%


VENTRICULAR ARRHYTHMIAS
NUMBER OF CASES
30
20
10
0
VPC'S
VT
VF
DEATH
ARRHYTHMIA
ANTERIOR
INFERIOR


TYPE OF INFARCT
INFERIOR
ANTERIOR

AGE:

AGE	NO.OF MALES	% OF MALES	NO. OF FEMALES	% OF FEMALES	TOTAL NO	%
30 - 39	12	14	-	-	12	12
40 - 49	14	16	03	21.5	17	17
50 – 59	23	27	06	43	29	29
60 – 69	23	27	02	14	25	25
70 - 79	13	15	03	21.5	16	16
80 - 89	01	01	-	-	1	1

TYPE OF INFARCT:

SEX	ANTERIOR	ANTERIOR	INFERIOR	INFERIOR
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
MALE	50	94	36	77
FEMALE	3	6	11	23

QT DISPERSION AMONG AMI:

QT dispersion in AMI was found to be significantly higher 82.56 ± 19.30 ms than in normal individuals 32.96 ± 10.06 ms, $p<0.001$. QT dispersion in patients of AMI was found to be higher on admission 82.56 ± 19.30 ms and was found to decrease later in the course of disease 62.12 ± 18.52 ms, 24 hours after admission and at the time of discharge from ICCU after an average stay of 7 days 51.98 ± 16.74 ms. The difference observed was statistically significant $p < 0.05$. The value at the time of discharge 51.98 ±16.74 ms was still higher than

normal 32.96 ± 10.06 ms and the difference was statistically significant $p < 0.05$. QT dispersion was significantly greater in patients with anterior wall MI 87.01 ± 18.16 ms than in those with inferior wall MI 77.53 ± 17.04 ms ; $p < 0.05$.

Type of infarct	**QT d (ms.)**
ANTERIOR	87.01 ± 18.16
INFERIOR	77.53 ± 17.04

QT DISPERSION AND ARRHYTHMIA:

Out of 100 patients of AMI, 49 developed ventricular arrhythmias. QT dispersion was significantly higher 98.77 ± 10.16 ms in 49 patients with ventricular arrhythmia than 51 patients without ventricular arrhythmias 66.98 ± 16.28; $p < 0.01$. 36 patients had ventricular premature beats(VPB's), 8 had ventricular tachycardia (VT) and 5 had ventricular fibrillation. QT dispersion was significantly higher in patients with VT/VF (124.61 ± 11.14 ms) than those who had only VPB's 89.44 ± 11.42 ; $p < 0.01$.

SEX	NO. WITH VPB'S	NO. WITH VT	NO. WITH VF
MALE	25	5	1
FEMALE	11	3	4

QT DISPERSION AND THROMBOLYTIC THERAPY:

Out of 100 patients of AMI, 58 were thrombolysed with streptokinase and 42 were not. No statistically significant difference was observed in QT dispersion in the two groups 51.75 ± 16.05 ms vs 52.28 ± 14.68 ms ; $p > 0.05$ at the time of discharge from ICCU.

QT DISPERSION AND PROGNOSIS:

Out of 100 patients of AMI, 6 patients died. 5 died due to ventricular arrhythmias and 1died due to causes other than arrhythmia. QT dispersion was found to be significantly higher in those who died 133.33 ± 10.97 ms than in those who survived 79.31 ± 17.91 ms ; $p < 0.05$. QT dispersion was significantly higher in arrhythmic deaths 134.4 ± 8.06 ms than in those who had non-arrhythmic deaths 128 ms ; $p < 0.05$.

AGE ADJUSTED MORTALITY RATES:

AGE GROUP	SEX	NO.OF CASES	DEATHS	% OF DEATHS
30-39	M	12	-	-
	F	-	-	-
40-49	M	14	-	-
	F	3	-	-
50-59	M	23	2	8
	F	6	1	16
60-69	M	23	-	-
	F	2	1	50
70-79	M	13	1	7
	F	3	1	33
80-89	M	1	-	-
	F	-	-	-

DISCUSSION

The present prospectively designed study aimed to examine QT dispersion in 100 patients of AMI and an equal number of age-and sex matched healthy individuals.

QT DISPERSION IN NORMAL INDIVIDUALS:

In normal individuals a low QT dispersion was observed 32.96 ± 10.06 ms. similar values of QT dispersion have been reported earlier[62-65]. Somewhat higher values have been reported in few other studies[66-68]. The results obtained by non-invasive assessment of QT dispersion from the surface echocardiogram are further substantiated by data obtained from endocardial or epicardial catheter mapping[69]. Using this method, several studies have demonstrated regional differences in ventricular repolarisation times of 40 to 55 ms[70-71]. Extensive body surface mapping has also been used to assess disparities in ventricular repolarisation in healthy persons and has revealed difference in QT duration of upto 60 ms[72]. Taken together these findings suggest that a range of QT dispersion between 30 and 50 ms appears to represent the normal limits of this parameter.

QT DISPERSION IN AMI:

QT dispersion in patients of AMI ranged from 40ms to 144ms with an average of 82.56 ± 19.30 ms which was significantly higher $p < 0.001$ than in normal healthy individuals 32.96 ± 10.06 ms. Patients with MI may have an inhomogenous ventricular repolarisation process. In the setting of AMI, the interplay between ischemic living tissue and relatively depolarized dying tissue would create a complex transition period affecting

QT interval dispersion. In early stage of AMI, increase in QT dispersion would be primarily due to local shortening of action potential. However within few hours prolongation of QT interval would become the dominant feature governing QT dispersion[73].

In AMI, QT dispersion was highest at the time of admission 82.56 ± 19.30 ms and was to decrease in the course of time, 62.12 ± 18.52 ms at 24 hrs after admission and 51.98 ± 16.74 ms at the time of discharge. The difference observed was statistically significant $p < 0.05$. Glancy et al[74] measured QTc dispersion on days 1,2,3 and 6 in 17 patients with AMI. They found the maximal QTc dispersion in the electrocardiogram taken on day 3. However in a large study of 316 consecutive patients, Newby et al [75] could not find significant difference in QT dispersion assessed at admission or after 2 and 3 days.

QT DISPERSION AND SITE OF INFARCT:

QT dispersion was significantly greater in anterior wall AMI 87.01 ± 18.16 ms than in inferior wall AMI 77.53 ± 17.04 ms ; $p < 0.05$. Similar observations have been made earlier[66-68]. However, Cowan et al[76] did not observe any significant difference in QT dispersion with different territory MI.

QT DISPERSION AND REPERFUSION THERAPY:

In essence, the determinant of increased QT dispersion during AMI are : speed of reperfusion, patency of the infarct related artery[IRA], and location of AMI. Quick restoration of blood in the IRA post-MI decreases QT dispersion. Studies have shown that post infarction patients with open arteries have a lower mortality rate than patients with closed arteries. Mortality rates as low as 2.5% have been reported in patients with patent

arteries compared with 15% in patients with closed arteries[77]. Mechanisms proposed to account for the beneficial effects of early and late reperfusion on mortality have been reviewed by Gersh and Anderson[78].

In the present study no statistically significant difference was noted in QT dispersion at the time of discharge from ICCU in those who received thrombolytic therapy 51.75 ±16.05 ms and those who did not 52.28 ± 14.68 ms ;$p > 0.05$. Some previous studies[68] also showed significant reduction in QT dispersion while others reported no change in QT dispersion after thrombolytic therapy[66,67].

QT DISPERSION, VENTRICULAR ARRHYTHMIAS AND MORTALITY IN AMI:

In experimental investigations, electrodes placed several millimeters apart with a small field of view have measured regional disparities in repolarisation. Variation in ventricular recovery time is an important factor in experimental tachyarrhythmias. The usual site of abnormal dispersion from which arrhythmias occur is at border zone of the infarcted area[79].

In the present study QT dispersion was significantly higher $p < 0.01$ in patients of AMI with ventricular arrhythmias 98.77 ± 10.16 ms than those without 66.98 ± 16.28 ms. QT dispersion was significantly higher $p < 0.01$ in those with VT/VF 124.61 ± 11.14 ms than in those with VPB's 89.44 ± 11.42 ms.

It simply illustrates the gradual increase in the heterogeneity of ventricular recovery from normal subjects to patients with uncomplicated MI to those with serious ventricular arrhythmias.

In the present study, QT dispersion at admission was high in patients with AMI who died than those who survived 133.33 ± 10.97 ms vs 79.31 ± 17.91 ms ;$p < 0.05$. QT dispersion at admission was higher in patients with AMI with arrhythmic death 134.4 ± 8.06 ms than those who had non-arrhythmic death 128 ms $p < 0.05$.

CONCLUSION

The conclusions derived from this study include

- Mean QT dispersion is significantly increased after Acute Myocardial Infarction.
- QT dispersion shows a dynamic decrease with time.
- Mean QT dispersion levels are higher in patients with ventricular tachycardia and ventricular fibrillation compared to patients with Acute Myocardial Infarction without these arrhythmias.
- The change in QT dispersion are dynamic, and it may serve as a non-invasive marker of susceptibility to malignant ventricular arrhythmias.
- Males outnumbered females in the study.
- Incidence is higher in older age group more so in females.
- Most of the patients had anterior wall infarction.

SUMMARY

As medical technology advances the physician is increasingly faced with a bewildering array of technical, diagnostic and therapeutic options for the treatment of Acute Myocardial Infarction. It has become important that the clinician be able to choose wisely among these available options, especially in the setting of Myocardial Infarction, where every minute is vital.

AMI is associated with changes in the electrophysiological properties of the heart. By means of assessing QT dispersion from the surface electrocardiogram there is convincing evidence that in AMI, inhomogeneity in ventricular repolarisation is augmented. Some clinical observations also indicate that this increased disparity in repolarisation is directly accompanied by the occurrence of ventricular arrhythmias and may help identify patients at high risk of sudden death. QT dispersion challenges our current approaches to the electrocardiographic assessment of arrhythmic risk. It provides a potentially simple, cheap, non-invasive method of measuring underlying dispersion of recovery of ventricular excitability.

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QT DISPERSION AND EARLY ARRHYTHMIC

RISK IN AMI

PROFORMA

Name: Age : Years. Sex: Male/Female
Occupation: I.P. no:
Address: D.O.A:
D.O.D:

DATE & TIME OF ONSET OF PAIN:

DATE & TIME OF ADMISSION:

SYMBTOMS: □ CHEST PAIN / □ DYSPNOEA / □ SWEATING / □ GIDDINESS

H/O DRUG INTAKE WHICH PROLONGS QT INTERVAL

FINAL DIAGNOSIS:

KILLIP CLASSIFICATION: I / II / III / IV

□ THROMBOLYSED □ NOT THROMBOLYSED

	ADMISSION	**DAY 2**	**AT DISCHARGE**
Pulse/min			
B.P. mmHg			
Basal crepts			
C.V.S.			

ELECTROCARDIOGRAPHIC FINDINGS:

DATE / DAY	**ON ADMISSION**	**24 HRS LATER**	**AT DISCHARGE**
RATE/MIN			
RHYTHM			
PR INTERVAL			
QRS DURATION			
ST SEGMENT			
T WAVE			
RR INTERVAL			
BLOCK			

QT DISPERSION			
ARRHYTHMIA			

ECHOCARDIOGRAPHIC FINDINGS:

CAHD-HYPOKINESIA OF ANTERIOR WALL

LOWER1/2 IVS

INFERIOR WALL

LVEF: %

OUTCOME:□ALIVE □DEATH □ NO ARRHYTHMIA □ ARRHYTHMIA- TYPE

MASTER CHART FOR HEALTHY CONTROLS

S.NO	NAME	AGE	SEX	QT d (ms)
1	YASHODHA	57	F	32
2	SUBBULAXMI	60	F	20
3	KARTHIKARANI	45	F	40
4	HELEN MARY	35	F	20
5	TAMILSELVI	40	F	32
6	RAJAMMAL	40	F	32
7	REITA	42	F	40
8	CHINTHAMANI	58	F	32
9	MOOKAYEE	65	F	40
10	VASANTHA	60	F	24
11	MAHALAXMI	63	F	44
12	MALLIGA BEGUM	52	F	32
13	AMSAVALLI	50	F	40
14	AROOVAM	62	F	24
15	NATARAJAN	60	M	40
16	AYYA	72	M	48
17	ASAI	75	M	36
18	SUNDARESAN	57	M	20
19	RAVI	31	M	32
20	CHINARASU	45	M	44
21	KARUPAIYAN	60	M	36
22	MURUGESAN	35	M	20
23	RAJAGOPAL	52	M	40
24	PANNEERSELVAM	50	M	36
25	KRISHNAMOORTHI	52	M	20
26	RAJENDIRAN	30	M	20
27	MURUGESAN	45	M	40
28	JAGANATHAN	50	M	36
29	RAMASAMY	63	M	48
30	THANDAYUTHAPANI	50	M	24
31	RAJENDIRAN	48	M	44
32	NALLAIYAN	55	M	32
33	KULANCHIAPPAN	40	M	40
34	NARASIMHAN	30	M	44
35	RAJA	30	M	20
36	DHANAPAL	45	M	28
37	CHANDIRAN	45	M	28
38	RAJENDIRAN	35	M	40
39	CHINNADURAI	32	M	44
40	CHINNAIYAN	78	M	40
41	KANNAN	67	M	24
42	PARAMASIVAM	55	M	40
43	VEERAMUTHU	56	M	36
44	NARAYANAMOORTHI	30	M	44
45	MANI	57	M	36
46	PACKIRISAMY	58	M	20
47	PARAMASIVAM	65	M	40
48	KALYAN	40	M	24
49	BASKARAN	57	M	48
50	MANI	45	M	32
51	SHANMUGAM	50	M	28

52	THANGARAJ	35	M	24
53	SELVARAJ	33	M	44
54	ANBALAGAN	43	M	24
55	RAMALINGAM	54	M	40
56	SUBRAMANIAN	33	M	20
57	THAVASI	45	M	32
58	KRISHNAN	66	M	36
59	GURUMOORTHI	45	M	24
60	MANIVEL	54	M	20
61	MARUTHAMUTHU	75	M	24
62	RAMU	50	M	36
63	RAMANATHAN	58	M	40
64	PERIYASAMY	34	M	40
65	RAJENDIRAN	39	M	24
66	KARUNANITHI	53	M	24
67	MURUGAIYAN	58	M	36
68	PALANIYANDI	65	M	48
69	KALIYAMOORTHI	55	M	36
70	GANESAN	49	M	40
71	LAKSHMAN	54	M	24
72	MURUGESAN	45	M	36
73	SHANKAR	35	M	44
74	JEYAKUMAR	46	M	36
75	SUNDARAMOORTHI	40	M	36
76	RAMALINGAM	45	M	40
77	RADHAKRISHNAN	40	M	44
78	KANNAN	48	M	48
79	DEVARAJ	43	M	40
80	GUNASEKARAN	50	M	32
81	MANICKAM	57	M	24
82	ANTONY SAMY	38	M	40
83	MD. ANIF	74	M	20
84	THIYAGARAJAN	60	M	36
85	PERIYASAMY	45	M	28
86	RAVICHANDRAN	30	M	24
87	SWAMINATHAN	63	M	36
88	SHANMUGANATHAN	65	M	40
89	RAVICHANDRAN	35	M	36
90	PANDIYAN	56	M	20
91	KRISHNAMOORTHI	43	M	28
92	SATHYAMOORTHI	76	M	40
93	KRISHNAMOORTHI	34	M	24
94	SOUNDARAJAN	45	M	24
95	RAJENDIREN	60	M	36
96	SAMINATHAN	50	M	24
97	SATHASIVAM	85	M	36
98	DURAISAMY	70	M	24
99	KULANTHAIVELU	60	M	20
100	AROCKIASAMY	62	M	20